

PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The THIRTY-FOURTH ANNUAL GENERAL MEETING of the British Cardiac Society was held at the Royal Infirmary, Manchester, on Thursday, May 26, 1955. The Chairman, CRIGHTON BRAMWELL, took the Chair at 9.30 a.m.; 126 members and 27 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual General Meeting, having been published in the Journal (16, 468, 1954) were taken as read and confirmed.
2. The balance-sheet for 1954-55 was presented, having been audited and found correct by A. Morgan Jones and E. Wyn Jones. The credit balance on May 9, 1955, was £1030 6s. 7d.
3. S. A. Levine was elected and acclaimed as an *Honorary Member* of the Society.
4. The following were elected *Extra-Ordinary Members*:

Arnold Stott	G. E. S. Ward
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5. The following Associate Members were elected as *Ordinary Members*:

R. I. S. Bayliss	R. Hudson
R. Byron Evans	P. Mounsey
J. D. Hay	E. G. Wade
B. B. Hosford	
6. The following *Surgical Members* were elected:

F. R. Edwards (Liverpool)	A. H. Siddons (London)
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7. The following new *Associate Members* were elected and introduced to the President and the Chairman:

D. W. Barritt (Bristol)	P. M. McAllen (London)
R. S. Duff (London)	T. Semple (Glasgow)
R. W. Emanuel (London)	T. B. Smiley (Belfast)
W. Lister (Plymouth)	(<i>Assoc. Surgical Member</i>)
A. C. MacDonald (Glasgow)	R. Steiner (London)
I. K. R. MacMillan (London)	R. S. Stevens (Worthing)
H. J. S. Matthew (Edinburgh)	O. L. Wade (Birmingham)
M. B. Matthews (Edinburgh)	
8. C. G. Baker and A. R. Gilchrist were elected members of the Council in place of P. H. Wood and J. H. Wright (term of office expired).
9. It was agreed that Rule 1 of the Society should be altered to read:

"The Society shall be called 'The British Cardiac Society'. Its objects shall be the advancement of knowledge of Diseases of the Heart and Circulation, and the promotion of good fellowship among those whose primary interest is in the practice of Cardiology, or in research in this or allied subjects."

It was also agreed that Rule 4 of the Society should be altered to read:

"There shall be a President of the Society who shall be nominated by the Council and elected by the Society. The Council shall give the Society at least one month's notice of the nomination, and if during that time any other nominations, which must be signed by ten Members, are received by the Council then the election shall be by ballot. He shall be elected for two years and may be eligible for re-election for further periods of one year to a total of five years on the recommendation of the Council. He shall be *ex officio* a member of the Council. He will represent the Society at home and abroad and may preside over meetings of the Council but not at the Scientific Meetings of the Society, for which a local Chairman will be elected annually."
10. The Secretary announced that the Council, at its previous meeting, had recommended that in future Associate Members should be allowed to attend the Private Business Session of Meetings of the Society,

provided it was decided that no good reason existed for their exclusion from any particular meeting, and the Secretary was instructed to circulate the Agenda of the Private Business to Associate Members in future.

SHORT COMMUNICATIONS

A CLASSIFICATION OF SYSTOLIC MURMURS

By AUBREY LEATHAM. Two varieties of systolic murmurs may be recognized, depending on their shape and relation to the heart sounds: (A) ejection murmurs, midsystolic and usually basal; and (B) regurgitant murmurs, pansystolic. Knowledge of their graphic configuration can be applied to clinical auscultation.

(A) Ejection systolic murmurs are midsystolic and are produced by turbulence from flow of blood through the aortic or pulmonary valves when there is stenosis, valvular damage without stenosis, dilatation of the vessel beyond the valve, or increased flow through the valve. There is an interval between the first sound and the onset of the murmur depending on the time taken by the ventricle to raise its pressure sufficiently to open the aortic or pulmonary valve. The flow and murmur begin as blood is ejected into the great vessels and rise to a crescendo about midsystole, diminishing as the ventricle relaxes and intra-ventricular pressure falls, and ceasing before the second sound produced by closure of the respective valve. Basal ejection vibrations can be recorded in many normal subjects and some slight increase in their intensity is probably responsible for most innocent murmurs.

(B) Regurgitant systolic murmurs are pansystolic and caused by flow of blood from a chamber or vessel that is at a higher pressure throughout systole than the receiving vessel or chamber. In mitral and tricuspid incompetence the ventricular pressure rapidly exceeds the atrial pressure and the backward flow and murmur begin very soon after the first sound. The flow and murmur continue right up to the second sound because the pressure difference between the two chambers is still great at the time of closure of the aortic and pulmonary valves, and tends to drown the second sound on clinical auscultation at the site of maximum intensity of the murmur. In some cases of mitral incompetence the murmur is maximal in late systole, but early systolic vibrations can always be recorded.

In ventricular septal defect with normal pressures the murmur is pansystolic because the left ventricular pressure is higher than the right throughout systole. In ventricular septal defect with high right-sided pressures and little left-to-right shunt, the pansystolic murmur is minimal or absent and there is an ejection midsystolic murmur from dilatation of the pulmonary artery. In patent ductus with normal pressures the left-to-right flow and murmur are continuous, and the murmur is obvious or maximal in late systole when an ejection systolic murmur would have diminished. With high pulmonary pressure and resistance and little or no left-to-right shunt the continuous murmur is replaced by a midsystolic ejection murmur from dilatation of the pulmonary artery. In two children with patent ductus and moderate elevation of pulmonary pressure and resistance but with obvious left-to-right shunt the pulmonary murmur was pansystolic presumably because the main left-to-right shunt was systolic.

In conclusion, it is hoped that the division of systolic murmurs into ejection midsystolic and regurgitant pansystolic murmurs will aid their identification and classification.

THE CLINICAL SYNDROMES OF PATENT DUCTUS WITH PULMONARY HYPERTENSION

By R. M. MARQUIS and A. RAE GILCHRIST. Twenty-six of a consecutive series of 252 patients with isolated patency of the ductus arteriosus did not conform to the typical pattern. Clinical recognition of the atypical case is usually possible, but the relative difficulty in diagnosis suggests that 10 per cent underestimates its incidence.

The 26 patients have three factors in common—absence of the continuous murmur, pulmonary hypertension, and the large size of ductus—but they form three distinct clinical groups that special investigations confirm.

Group I (8 cases, 2 operations, 3 autopsies). Pulmonary hypertension, secondary to large left-to-right shunt, sometimes sufficient to cause partial shunt reversal.

Group II (9 cases, 8 operations, 3 autopsies). Pulmonary hypertension, early in onset, initially sufficient to restrict the left-to-right shunt and probably advancing to shunt reversal.

Group III (9 cases, no operations, 2 autopsies). Pulmonary hypertension primary with permanent shunt reversal.

Peripheral cyanosis may be present in all groups. In the presence of shunt reversal central cyanosis may be generalized, dissociated, or only detectable on analysis of arterial blood distal to the ductus.

Some patients with continuous murmurs and large left-to-right shunts pass into Group I but Groups II and III never have continuous murmurs. In Group III pulmonary hypertension is the primary dysfunction and surgical occlusion of the ductus is probably unwise. Early surgery may be curative in Groups I and II.

COMPARATIVE STUDY OF THE RADIOLOGICAL KINETICS AND THE ARTERIAL BLOOD PRESSURE IN CONGENITAL HEART DISEASE

By E. DONZELOT, V. SERAFINI, J. BOUGNOUX, P. KERVoeLEN, and R. HEIM DE BALSAC. To be published in full: *Brit. Heart J.*, 18, January 1956.

THE INTRACARDIAC ELECTROGRAM AS AN AID IN LOCALIZATION OF PULMONARY STENOSIS

By D. EMSLIE-SMITH and K. G. LOWE (*introduced by I. G. W. Hill*). To be published in full: *Brit. Heart J.*, 18, January 1956.

AN EVALUATION OF LEAD V4R IN THE CARDIOGRAPHIC DIAGNOSIS OF RIGHT VENTRICULAR HYPERTROPHY

By F. CAMERINI (*introduced*), J. F. GOODWIN, and MAX ZOEB. To be published in full: *Brit. Heart J.*, 18, January 1956.

THE POSTCOMMISSUROTOMY SYNDROME

By CORNELIO PAPP and M. M. ZION (*introduced*). In 22 of 105 patients (21%) who had mitral valvotomy, the immediate or late postoperative period was complicated by the postcommissurotomy syndrome (*P.C.S.*). The main symptoms were pyrexia, pericarditis, left pleural effusion, and a tendency to relapse. The *P.C.S.* has been attributed to and confused with postoperative rheumatic activity because of pericardial involvement, pyrexia, the tendency to relapse, and the increased sedimentation rate—common features to both. However, preceding streptococcal infection, joint swellings, myocardial and valvular involvement, and the effect of salicylate are the distinguishing signs of rheumatic disease and these are absent in the *P.C.S.* These two entities, therefore, should be clearly separated.

Pyrexia for a few days, left pleural effusion that absorbs within a fortnight, and fleeting signs of pericarditis are the accompanying signs of the early postoperative period in the uncomplicated cases. The *P.C.S.* is the accentuation, prolongation, and recurrence of these normal postoperative sequelæ rather than a superadded disease. The causes for persistence and relapse are local and not general. The effusion was found to be bloodstained in 9 out of 11 cases after the first week when in the controls without *P.C.S.* it was found to be serous. In addition, loculus formation was observed in four; aspiration of the loculi led to prompt recovery. Postoperative oozing from the auricular wound into the pleuro-pericardial space may be the main cause of the syndrome. Loculus formation and pulmonary consolidation beneath the effusion may represent added factors.

The *P.C.S.* has an invariably good prognosis—a feature also at variance with rheumatic fever—and has no adverse effect on the outcome of the operation.

THE BALLISTOCARDIOGRAM AND THE PULMONARY ARTERY PRESSURE IN MITRAL STENOSIS

By C. B. HENDERSON. The ballistocardiograms of 100 cases of mitral stenosis have consistently shown two abnormal waves. These waves are present in auricular fibrillation and in sinus rhythm and are named the RI and RJ waves. The RI wave is a footward deflection commencing 0.06–0.10 sec. after the Q wave of the electrocardiogram and the RJ wave is a headward deflection immediately following the RI wave.

The abnormality can be divided into five grades of severity. The grade varies with changes in pulmonary artery pressure as when the heart rate changes, and the duration of the preceding diastolic pause varies, with the changing phases of respiration, and with the improvement following successful mitral valvotomy. The higher the pulmonary artery pressure the higher is the grade of abnormality of the ballistocardiogram.

Evidence is presented chiefly in the form of ballistocardiograms and pressure tracings from the pulmonary artery recorded simultaneously to suggest that the abnormal waves are a result of right ventricular ejection. A close correlation is shown between the grade of abnormality of the ballistocardiogram and the pulmonary artery pressure in the 100 cases investigated. A prediction of the mean pulmonary artery pressure can be made in patients with mitral stenosis from the ballistocardiogram and in ten cases this was confirmed at subsequent catheterization.

THE QUANTITATIVE ESTIMATION OF VALVULAR REGURGITATION BY DYE DILUTION CURVES

By PAUL KORNER (*introduced*) and JOHN SHILLINGFORD. *Illustrated by a colour film.* In normal dye dilution curves the shape of the curve can be accurately predicted for any given cardiac output and volume between the site of injection and sampling point. The introduction of valvular incompetence causes a deviation from this normal pattern and the amount of this deviation is related to the amount of phasic regurgitant flow.

The validity of the hypothesis has been tested in a heart circulation model and found to have a high degree of accuracy. A standard nomogram has been calibrated from normal model curves and has been used for predicting the phasic regurgitant flow in the model.

A separate and independent nomogram has been calculated for man for the estimate of regurgitant flow. This may be estimated either for the left or right sides of the heart at the time of cardiac catheterization by injecting the dye either into the pulmonary artery or into the right atrium.

Examples of the amount of phasic regurgitant flow in different valve lesions are shown and discussed.

The film shows the behaviour of the passage of dye through the heart model with and without valve incompetence and the application of the technique to man.

OBSERVATIONS ON THE HISTORY OF THE PICTORIAL REPRESENTATION OF THE HEART

By A. SCHOTT. The paper attempts to describe briefly some of the more important stages in the historical development of the pictorial representation of the heart. Starting with the *Fasciculus Medicinæ* of Johannes de Ketham of 1491, it includes references to Leonardo da Vinci, Vesalius, Eustachius, Fabricius, Lower, Haller, and Scarpa. The most recent is Quain as representative of the middle of last century. The paper is illustrated by slides.

EFFECTS OF SQUATTING

By E. P. SHARPEY-SCHAFFER. In normal subjects arterial mean and pulse pressure increased on squatting and there was vasodilatation of forearm vessels. Cases of heart failure showed no increase of pulse pressure or vasodilatation. On standing upright after squatting there was decrease of pulse pressure and constriction in normal subjects but not in cases of heart failure.

The results support the theory of baroreceptor responses to acute pulse pressure changes.

TOTAL AND PERIPHERAL FLOW IN ACUTE MYOCARDIAL INFARCTION

By G. DE J. LEE (*introduced by E. P. Sharpey-Schafer.*) Eleven patients admitted to hospital with acute myocardial infarction were studied; two subsequently died as a result of their condition. Pulse rate, blood pressure, central venous pressure, cardiac output (Hamilton dye technique) and forearm bloodflow (venous occlusion plethysmography) were obtained. The observations were repeated after 4-6 weeks in those who recovered.

The central venous pressure was raised initially. Pulse rate varied between individuals, but tended to decrease with recovery. The initial pulse rate did not correlate well with the apparent severity of the infarct. Peripheral vasoconstriction, indicated by low forearm flow, was also most marked in the severe cases, and in some instances it was intense: this was sometimes associated with little fall in blood pressure.

Cardiac output in three cases without shock was normal in one and raised in two. In the remainder, the cardiac output was below normal and tended to be more reduced the severer the infarct. However, the cardiac output was not as low as in a separate group of patients with hypertensive cardiac failure. In the two patients who died from their infarcts, the cardiac output was 5.0 and 5.7 litres a minute respectively. Central blood volume was raised in all instances initially and returned to normal with recovery. This increase in central blood volume was less than in the group of patients with hypertensive heart failure.

CONTROL OF QUINIDINE DOSAGE BY BLOOD AND URINE ESTIMATIONS

By D. WEITZMAN (*introduced by Graham Hayward*). Published in full: *Brit. Heart J.*, 17, 534, 1955.

THE EFFECTS OF PERFUSION AND CONGESTION OF THE PULMONARY CIRCULATION ON THE DISTENSIBILITY OF THE RABBIT'S LUNG

By PETER HARRIS (*introduced by Samuel Oram*). In these experiments the lungs of rabbits have been removed from the thorax and distended by positive pressure with air. Air has then been withdrawn from the lungs in small measured successive amounts and the intratracheal pressure recorded at each stage. In this way the relationship between pressure and volume in a distended lung can be determined and this relationship can be expressed in a graph. The curve that is derived is the static pressure-volume curve of the lungs, since the effects of viscous resistance to flow of air are eliminated and only the force of elastic recoil of the lungs is operating.

When the pulmonary circulation is perfused or congested in various ways, the static pressure-volume curve of the lungs is altered. An analysis of such curves leads to the following conclusions.

(1) The effects of perfusion or congestion of the pulmonary circulation on the static pressure-volume curve of the lungs are due entirely to alterations in the capillary circulation. The distension of arteries and veins has in itself no substantial mechanical influence on the lungs.

(2) Congestion of the pulmonary capillaries imposes a force on the lungs that tends to maintain them in a position of partial inflation.

(3) Congestion of the pulmonary capillaries also causes them to occupy a small measurable part of the alveolar air space.

The effects described may be applicable to the mechanical alteration of the lungs found in diseases of the mitral valve and in failure of the left ventricle.

REACTIVITY OF PERIPHERAL BLOOD VESSELS TO ADRENALINE AND NORADRENALINE IN HUMAN HYPERTENSION

By ROBERT S. DUFF (*introduced by Geoffrey Bourne*). A method of measuring precisely, under controlled conditions, the purely local effects of minimal amounts of drugs on the calibre of blood vessels in the extremities is presented. Repeated determinations of the volume of blood flowing through portions of the limbs are made by venous occlusion plethysmography; at regular intervals solutions of drugs alternating with plain saline are introduced into the artery supplying the tissue during the course of a continuous, constant-rate, intra-arterial infusion.

Quantitative estimates of the peripheral vascular effects of very low concentrations of adrenaline and noradrenaline have been compared in various categories of patients with normal and raised blood pressure. A proportion of patients with raised blood pressure has been found to have increased sensitivity to one or both agents. Aetiological and therapeutic implications of this finding are discussed.

ELECTROCARDIOGRAPHIC CHANGES IN EXPERIMENTAL HYPOTHERMIA

By GERALD R. GRAHAM (*introduced by W. Somerville*). Ventricular fibrillation is one of the main problems in artificial hypothermia for intracardiac surgery. Its incidence and mode of onset, under various circumstances, are still unsettled. Also, it is not clearly established whether warning signs, in the form of arrhythmias or electrocardiographic pattern changes, regularly occur before its onset.

To investigate these points, experiments were performed on anaesthetized dogs, maintained under artificial respiration but not otherwise operated on, that were cooled to 25° C. In one series, hypothermia was induced by extracorporeal circulation through a cooling coil; in the other, cooling was achieved by placing the dogs in an ice-bath. In both, the dogs were re-warmed over a period of a few hours. Intra-arterial blood pressure was recorded and continuous electrocardiographic control maintained (limb and chest leads), permanent records being taken at frequent intervals.

It was found that when ventricular fibrillation occurred it almost always did so without previous warning (arrhythmias or other electrocardiographic changes) that could not also be observed in other animals without the onset of ventricular fibrillation. QRS and S-T-T changes were principally related to the cooling effects *per se* and were reversible. These abnormalities seemed less marked with bath cooling. Typical cardiographic changes and their reversibility were presented and their significance discussed.